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# Is particulate air pollution associated with health and health inequalities in New Zealand?

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# **Is particulate air pollution associated with health and health inequalities in New Zealand?**

## **ABSTRACT**

Air pollution can increase mortality risk and may also exacerbate socioeconomic inequalities in health outcomes. This New Zealand study investigated whether exposure to particulate air pollution (PM<sub>10</sub>) was associated with mortality and health inequalities. Annual mean PM<sub>10</sub> estimates for urban Census Area Units (CAUs) were linked to cause-specific mortality data. A dose-response relationship was found between PM<sub>10</sub> and respiratory disease mortality, including at concentrations below the existing annual average guideline value of 20 µg m<sup>-3</sup>. Establishing and enforcing a lower guideline value is likely to have population health benefits. However, socioeconomic inequalities in respiratory disease mortality were not significantly elevated with PM<sub>10</sub> exposure.

## **KEY WORDS**

Air pollution, New Zealand, mortality, respiratory disease, health inequalities

## INTRODUCTION

The numerous health effects of exposure to ambient air pollution have been well documented (Brunekreef and Holgate, 2002; Stieb et al., 2002). In particular the health effects of airborne particulate matter have received much attention, especially those with an aerodynamic diameter  $< 10 \mu\text{m}$  (known as  $\text{PM}_{10}$ ), which can be inhaled into and deposited in the lungs (Nel, 2005). Adverse health outcomes associated with increased  $\text{PM}_{10}$  exposure include mortality, and some of the largest effects have been found for deaths from respiratory disease (Levy et al., 2000; Schwartz, 1994; Stieb et al., 2002; World Health Organization, 2004b). Increased mortality has been associated with both acute and chronic exposure to  $\text{PM}_{10}$  (World Health Organization, 2004a).

Health outcomes are often socially patterned (Mackenbach et al., 2003), including those with an aetiological link with exposure to particulate pollution. Significant inequalities in health outcomes exist between advantaged and disadvantaged communities in many countries, and the gaps are widening (Mackenbach et al., 2003; Pearce and Dorling, 2006). Strong socioeconomic gradients have been found for causes of death linked to air pollution, such as cardiovascular disease, respiratory disease and lung cancer (Huisman et al., 2005; Mackenbach et al., 2003; Prescott et al., 2003), with deprived groups consistently suffering worse health. Reducing socioeconomic health inequalities is a priority for many regional and national administrations. Identifying how inequalities can be reduced necessarily involves identifying the underlying factors.

Whilst numerous studies have described significant socioeconomic inequalities in particulate pollution-related health outcomes, the precise mechanisms that underlie these observations are poorly understood. There is evidence that air pollution exerts an effect on health to a

greater extent among those of low socioeconomic status (SES) (Laurent et al., 2007; O'Neill et al., 2003). Studies from certain cities in Canada (Jerrett et al., 2004), Norway (Naess et al., 2007), Brazil (Martins et al., 2004) and the United States (Krewski et al., 2003) have demonstrated that socioeconomic status modifies the relationship between air pollution and health outcomes in these regions.

Two potential mechanisms by which the environment may disproportionately affect low SES populations, and therefore influence health gradients, have been proposed: (i) disproportionate exposure of low socioeconomic status populations to pathogenic environments and/or (ii) increased susceptibility of low socioeconomic status populations to pathogenic environments (Laurent et al., 2007; O'Neill et al., 2003). Whether disadvantaged populations are disproportionately exposed to poor environments has been widely studied, using the framework of environmental justice. There is consistent evidence that low income and/or ethnic minority communities are exposed to higher levels of a range of air pollutants in many countries (Ito and Thurston, 1996; Jerrett et al., 2001; Mitchell and Dorling, 2003; Pearce and Kingham, 2008; Perlin et al., 2001). The frequently-observed spatial coincidence of environmental risks in communities of low socioeconomic status has been termed a 'double jeopardy' (US Institute of Medicine, 1999). In addition, disadvantaged populations may be more susceptible to the adverse effects of air pollution because of the greater prevalence of social and behavioural determinants of poor health often found in these populations. Such determinants include pre-existing poorer health status (e.g., diabetes, obesity) and health behaviours (e.g., diet, smoking), greater psychosocial stress (e.g., financial strain, lack of control over one's life) and poorer healthcare (e.g., accessibility, quality) (Laurent et al., 2007).

The focus of this study is New Zealand where poor air quality is recognised as an important public health challenge (Scoggins et al., 2004). The primary national policy approach has been to introduce ambient air quality guideline values, under the Resource Management Act 1991, which are enforced by regional, city or district councils (Ministry for the Environment, 2002, 2005). Guideline values are intended to represent the maximum concentration acceptable for the protection of human health and the environment (Ministry for the Environment, 2002). For PM<sub>10</sub>, guideline values are specified for exposures in both the short term (daily mean of 50 µg m<sup>-3</sup>) and the long term (annual mean of 20 µg m<sup>-3</sup>). The daily standard is frequently exceeded in cities in winter because of solid fuel-burning domestic heating systems (Krivácsy et al., 2006; Spronken-Smith et al., 2002). In the South Island city of Christchurch, short-term elevations in PM<sub>10</sub> have been associated with increases in a range of health endpoints including biomarkers, school absences, hospital admissions, respiratory symptoms and mortality (Cavanagh et al., 2006; Epton et al., 2008; Fisher et al., 2007; Hales et al., 2000; McGowan et al., 2002; Wilson et al., 2010). Long-term PM<sub>10</sub> exposure has also been linked with increased mortality risk in Auckland (Shrestha, unpublished PhD thesis, cited in Fisher et al., 2007). However, air pollution monitoring inconsistencies across the country have precluded quantification of the health risks associated with PM<sub>10</sub> exposure (whether long-term or short-term) at the national level.

Social and spatial inequalities in health outcomes have widened markedly in New Zealand since the 1980s and show little sign of narrowing (Blakely et al., 2005a; Pearce and Dorling, 2006; Pearce et al., 2008; Shaw et al., 2005). The gap between areas of high and low social deprivation has widened for a range of health outcomes, including those that have been associated with particulate air pollution such as cardiovascular and respiratory diseases (Pearce et al., 2008). However, whether the health of disadvantaged populations in New

Zealand is disproportionately affected by particulate air pollution has not received attention. It is therefore unclear whether particulate air pollution may have contributed to the widening health inequalities observed. Pearce and Kingham (2008) found evidence for greater exposure to particulate air pollution amongst low SES communities in New Zealand, and called for investigation into the implications of this social patterning for health and health inequalities.

In the current study, we utilised a recently developed measure of particulate air pollution for small areas across urban parts of New Zealand (Kingham et al., 2008). We were therefore able to address the following research questions for the urban population of New Zealand:

1. Is  $PM_{10}$  associated with cause-specific mortality?
2. Is the health of low SES populations disproportionately affected by  $PM_{10}$ ?

## **MATERIALS AND METHODS**

We selected small areas across urban parts of New Zealand for which particulate air pollution data were available. The pollution data were then joined with mortality records to investigate whether exposure to  $PM_{10}$  at the small area level was associated with specific causes of death. We also investigated whether the health differences between affluent and deprived areas increased with exposure to  $PM_{10}$ , as hypothesised.

### **Geographical unit of analysis**

Census Area Units (CAUs) were used as our small area geography for the analysis. CAUs are the second smallest census geography in New Zealand, and the smallest areal unit for which

mortality data are routinely disseminated. Of the 1842 CAUs in New Zealand we selected all urban CAUs for which PM<sub>10</sub> estimates were available ( $n = 970$ ). These CAUs had a mean area of 3.9 km<sup>2</sup> (range 0.1 to 301) and a mean population in 2001 of 2862 (range 3 to 8820).

### **Air pollution measure**

For urban CAUs across New Zealand (71% of the population; Statistics New Zealand, 2004) we obtained estimates of annual average PM<sub>10</sub> concentration from a validated land use regression model that has been described elsewhere (Kingham et al., 2008). The model was developed from an atmospheric dispersion model (Zawar-Reza et al., 2005) that combined meteorological and emissions data to produce reliable estimates of CAU-level PM<sub>10</sub> concentrations for the city of Christchurch. For the national model a range of nationally-available variables were used as proxies for particulate pollution emissions by source (domestic wood combustion, industrial and vehicular). For domestic heating the variables used were: density of wood fires (from a question relating to home heating in the census); number of winter days where the temperature fell below 5°C, mean wind speed was below 3 m s<sup>-1</sup> and there was no rainfall (conditions likely to result in high pollution levels); and an estimated area-level wood-use factor (an indication of wood use or availability derived from a government survey). For vehicular emissions the variables used were: vehicle kilometres travelled, the number of CAUs within 5 km of the CAU centroid (a surrogate for relative proximity to central city) and the number of days where mean wind speed was below 3 m s<sup>-1</sup> and there was no rainfall. Industrial emissions were predicted using local government emissions data for major industry and population density for local minor sources. Background levels of PM<sub>10</sub> (which derive from windblown dust and sea salt) were estimated based on local topographic characteristics and expert advice.



To validate the model the estimated PM<sub>10</sub> concentrations were compared against monitored data where available, revealing a high level of agreement ( $R^2 = 0.86$ ). The model performed equally well in high and low pollution areas and predicted slightly better in South Island ( $R^2 = 0.88$ ) than in North Island ( $R^2 = 0.76$ ). The disparity may have arisen partly because of the model had been partly based on statistical relationships between modelled and monitored data for Christchurch. An extract of the dataset is shown for Christchurch in Figure 1.

[Figure 1]

For this health risk analysis we partitioned the CAUs into low, medium and high PM<sub>10</sub> groupings. Given the annual average guideline value of  $20 \mu\text{g m}^{-3}$  (considered the maximum acceptable level for health) we set one break point at  $20 \mu\text{g m}^{-3}$  and split the larger fraction into equal halves (by number of CAUs). The resulting groups were therefore  $5.3$  to  $13.8 \mu\text{g m}^{-3}$  (low PM<sub>10</sub>),  $13.9$  to  $20.0 \mu\text{g m}^{-3}$  (medium PM<sub>10</sub>) and  $20.1$  to  $32.9 \mu\text{g m}^{-3}$  (high PM<sub>10</sub>).

## **Health data**

We selected a prevalent health outcome that, based on the international and New Zealand literature, was plausibly associated with particulate air pollution. There is clear evidence that exposure to PM<sub>10</sub> increases the risks of mortality from respiratory disease (Abbey et al., 1999; Stieb et al., 2002; World Health Organization, 2004b). To act as a control, for which we would not expect to find an association with PM<sub>10</sub>, we selected an additional prevalent health outcome for which air pollution has no apparent aetiologic role: colorectal cancer. The existence of a socioeconomic mortality gradient is well established for respiratory disease (Kunst et al., 1998), as well as for colorectal cancer (Auvinen, 1992; Steenland et al., 2002).

Individual-level mortality records (including age, sex, domicile of residence, and year of death) for the period 2001 to 2005 were obtained from the New Zealand Ministry of Health, and matched to CAUs. We extracted all deaths from respiratory disease (ICD-9 460-519; ICD-10 J00-J99) and colorectal cancer (ICD-9 153-154; ICD-10 C18-C20). Cause-specific mortality counts were generated by sex, age-group (0-15, 15-34, 35-54, and 55-74) and CAU. The analysis was restricted to persons under 75 in order to study premature mortality. Denominator age-group and sex-specific population counts were obtained for each CAU and study year. The total study population was approximately 2.8 million in 2001. Between 2001 and 2005, deaths from respiratory disease and colorectal cancer totalled 9115 and 4191, respectively.

We extracted area-level socioeconomic deprivation scores for the CAUs using the New Zealand Deprivation Index (NZDep2001) (Salmond and Crampton, 2002), and calculated deprivation quartiles for the urban CAUs in our sample. NZDep2001 is an area-level measure of deprivation that has been widely used in epidemiological studies (e.g., Mantell et al., 2004; Witten et al., 2008). The measure was created by combining nine weighted area-level census variables (e.g., income and employment status) (Salmond and Crampton, 2002).

### **Covariates**

Only age and sex were available for the individuals in the mortality dataset. To account for the potentially confounding influence of ethnicity and smoking behaviour (Blakely et al., 2005b; Miller et al., 2007; Pope et al., 2002) we derived area level measures of each. For each CAU we used census data to calculate percentage Māori and Pacific Islanders combined (in 2001) and mean percentage regular smokers (in 1996 and 2006).

The models were not adjusted for co-occurring pollutants (e.g., carbon monoxide or nitrogen dioxide), because previous work in New Zealand had found that multi-collinearity of these pollutants obscured joint models, and that the strongest health associations tended to be found for PM<sub>10</sub> (Fisher et al., 2007). To guard against auto-correlation we opted not to adjust for climate, because this was one of the variables used to estimate PM<sub>10</sub> levels.

## **Analyses**

We used regression analyses to investigate whether each cause of mortality was associated with PM<sub>10</sub> air pollution. Over-dispersion of the mortality count data made Poisson models unsuitable, hence negative binomial regression models were used (Hilbe, 2007). After running a baseline model for the relationship between PM<sub>10</sub> and mortality (adjusting for only individual age group and sex), relevant confounders were entered sequentially: area deprivation quartiles in model 2; area ethnicity quartiles in model 3; and area smoking rate quartiles in model 4. The population of each age-sex group in each CAU was entered as the exposure variable, and the models utilised robust standard errors to allow for spatial clustering (Williams, 2000). Incidence rate ratios (IRRs) and 95% confidence intervals (CIs) were calculated for medium and high PM<sub>10</sub> CAUs, relative to low PM<sub>10</sub> CAUs. Using continuous area deprivation, ethnicity and smoking rate variables in the models instead of quartiles made no substantive difference to the results, hence are not reported.

We subsequently investigated whether exposure to PM<sub>10</sub> acts to accentuate socioeconomic health inequalities. We stratified our regression models by PM<sub>10</sub> level in order to quantify the deprivation-related health disparity in groups of CAUs exposed to approximately the same level of particulate air pollution. Interactions were used as a formal test of whether the

association between socioeconomic deprivation and mortality varied by exposure to PM<sub>10</sub>. Significance of the resulting coefficients was tested using a Wald test.

## RESULTS

After omitting urban CAUs with incomplete data (whether air pollution, health outcomes or confounders,  $n = 7$ ) our sample contained 963 CAUs with an average population in 2001 of 2927.

### Is PM<sub>10</sub> exposure associated with mortality?

Particulate air pollution was positively associated with respiratory disease mortality (Table 1). Compared with CAUs exposed to the lowest PM<sub>10</sub> concentrations, respiratory disease mortality risk was significantly elevated at both medium and high levels (IRRs 1.17 and 1.26 respectively). The effect sizes revealed by the baseline model were attenuated slightly after adjustment for area deprivation (model 2), but were strengthened after additional control for area ethnicity (model 3). Adjustment for area-level smoking rate attenuated the IRR for the most polluted CAUs from 1.28 to 1.18. Thus, after full adjustment, CAUs exposed to the highest levels of PM<sub>10</sub> had an 18% greater risk (95% CI 3 to 35%) of respiratory disease mortality than those exposed to the least PM<sub>10</sub>. The IRRs associated with area deprivation and smoking rates were higher, suggesting that these are stronger determinants of respiratory disease mortality than particulate air pollution. A clear dose-response trend was seen (model 4 incidence rate ratio (IRR) for each increase in PM<sub>10</sub> level = 1.10, 95% confidence interval (CI) 1.03 to 1.18,  $p$  trend = 0.004). As anticipated, no significant relationship with PM<sub>10</sub> was identified for colorectal cancer mortality (Table 2).

[Tables 1 and 2]

### **Does exposure to PM<sub>10</sub> increase health inequalities?**

We used stratified models to examine the relative socioeconomic health inequality within groups of CAUs exposed to approximately the same level of PM<sub>10</sub>. The analysis was restricted to respiratory disease mortality because, as anticipated, we did not find a relationship between colorectal cancer and PM<sub>10</sub> levels in the fully specified model. The IRRs are presented relative to the least deprived CAUs at the same level of PM<sub>10</sub> exposure (Figure 2).

We found a clear socioeconomic gradient in respiratory disease mortality (Figure 2). At each level of PM<sub>10</sub> exposure, risk of respiratory disease mortality increased with increasing socioeconomic deprivation, after control for relevant individual- and area-level covariates. We found significant inequality, with the most deprived CAUs having over 1.7 times the risk of respiratory disease mortality than the most affluent areas.

The inequality was slightly more pronounced in populations with the highest level of PM<sub>10</sub> exposure. Wide confidence intervals, however, rendered this inequality not significantly different from that observed in the least polluted CAUs (most polluted: IRR 2.51, 95% CI 1.27 to 4.97; least polluted: IRR 2.11, 95% CI 1.46 to 3.05). An interaction model confirmed that the PM<sub>10</sub> and respiratory disease mortality relationship was not significantly modified by area-level deprivation (Wald test  $\chi^2 = 5.07$ ,  $p = 0.53$ ).

[Figure 2]

## DISCUSSION

Air pollution has well established links with increased mortality risks in a number of countries, and has been suggested as a potential driver of socioeconomic inequalities in pollution-related health outcomes. Identification of the factors that underpin poor health and health inequalities in various societies is a matter of considerable importance, yet little national air pollution and health research has previously been undertaken in New Zealand. This study investigated whether local variations in exposure to particulate air pollution ( $PM_{10}$ ) were associated with mortality and socioeconomic health inequalities in urban areas of New Zealand, using a national validated pollution dataset.

We found that  $PM_{10}$  had a clear dose-response relationship with respiratory disease mortality: populations with medium ( $13.9 - 20.0 \mu g m^{-3}$ ) and high ( $> 20.0 \mu g m^{-3}$ )  $PM_{10}$  exposure had significantly elevated risks of 15% and 18% respectively, compared with the least exposed CAUs ( $< 13.9 \mu g m^{-3}$ ). Area deprivation and smoking rate were also associated with respiratory disease mortality and therefore were potential confounding factors. However, adjustment for these variables only slightly reduced the risk attributable to  $PM_{10}$ , suggesting that the association of respiratory mortality with  $PM_{10}$  levels was not due to confounding by these factors.

Similarly, Jerrett et al. (2005) found that socioeconomic covariates reduced but did not eliminate the relationship between particulate air pollution and mortality. Significant respiratory disease mortality risks have also been associated with  $PM_{10}$  in many other countries, including those in Europe, the Americas and Asia (e.g., Abbey et al., 1999; Stieb et al., 2002; World Health Organization, 2004b). In New Zealand,  $PM_{10}$  has been associated with increased respiratory disease mortality in Christchurch (4% increase for a  $10 \mu g m^{-3}$

increase in daily PM<sub>10</sub>; Hales et al., 2000) and Auckland (33% increase for a 10 µg m<sup>-3</sup> increase in annual PM<sub>10</sub>; Fisher et al., 2007), but ours is the first study to have examined the relationship at the national level. By conducting a nationwide analysis we have shown that these significant health associations of PM<sub>10</sub> are generalisable for other urban parts of the country. It is therefore reasonable to expect that a modest reduction in annual mean PM<sub>10</sub> could have noteworthy health benefits for New Zealand, particularly in terms of respiratory health. Our findings add further evidence to the international literature linking particulate air pollution and respiratory health and demonstrate that health effects can be detected even in countries with relatively low levels of particulate pollution.

Moreover, the work has important implications for policy makers, as ours is the first study to have specifically investigated whether areas meeting the national health-based guideline value of 20 µg m<sup>-3</sup> are protected from adverse health effects. Compared with the least polluted CAUs, those experiencing annual average PM<sub>10</sub> concentrations of 13.9 to 20.0 µg m<sup>-3</sup> had a significantly greater risk of respiratory disease mortality, and the excess risk was only marginally lower than that found for the most polluted CAUs (15% compared with 18%). So the national guideline value, which is considered to set an acceptable annual level for PM<sub>10</sub> (Ministry for the Environment, 2002), is not sufficiently low to protect human health. Our findings concur with studies from other countries that have reported air pollution health effects at or below the existing health guidelines (e.g., Barnett et al., 2006; Zmirou et al., 1998). Indeed, the existence of ‘no-effect threshold’ values is now doubted (World Health Organization, 2004a). Our results suggest that establishing and enforcing a lower guideline value would have health benefits for the New Zealand population. In future work we will estimate the population health benefits of meeting various hypothetical lower guideline values.

Pearce et al. (Pearce and Kingham, 2008; Pearce et al., 2006) reported a social gradient in  $PM_{10}$  exposure across the CAUs in our study, following adjustment for the ethnic composition of the neighbourhoods (CAUs). They postulated that this environmental inequality may help explain inequalities in pollution-related health outcomes. We therefore investigated whether increased exposure to  $PM_{10}$  was associated with increased socioeconomic inequality in respiratory disease mortality. The results indicated that socioeconomic inequalities were slightly greater in the most polluted communities, compared with the least polluted, although the increase was not statistically significant. Research in other countries (reviewed by Laurent et al., 2007) suggests that the more deprived tend to experience greater health effects of air pollution. Our findings suggest that this heightened vulnerability, if present, is less pronounced in New Zealand, as we found little evidence that health inequalities were accentuated in high exposure communities. Other aspects of low socioeconomic status (e.g., poverty, poor housing) are therefore likely to be more important in explaining health inequalities than particulate air pollution.

A number of limitations of the work must be borne in mind. First, the study was cross-sectional and hence a causal relationship between air pollution and respiratory disease mortality cannot be assumed. Nonetheless, our inclusion of a cause of death for which there is no evidence of an aetiological role of particulate air pollution (colorectal cancer) helped provide some evidence that the respiratory disease effect was environmentally influenced.

Second, misclassification of exposure to  $PM_{10}$  was possible because an individual's exposure to air pollution will be influenced by factors such as their mobility into other neighbourhoods (e.g., travelling to work or school), and time spent indoors. The  $PM_{10}$  concentration data we used were based on fixed outdoor sites and hence assumed individual exposure was static and



outdoors. The data were also estimates rather than measurements for each CAU, hence model accuracy was a possible limitation. Nonetheless, we utilised spatially detailed measures of particulate pollution whereas previous studies have tended to use spatially coarse measures. The model was also validated and found to provide reliable PM<sub>10</sub> estimates for Christchurch. The approach used in the current paper is therefore a more precise reflection of intraurban variations in exposure to ambient PM<sub>10</sub> (Kingham et al., 2008; Wilson et al., 2005). Nonetheless, other measures of PM<sub>10</sub> exposure with finer *temporal* resolution (e.g., maximum concentrations, or numbers of threshold exceedances) might be more closely related to health. The sensitivity of the relationship to the specific PM<sub>10</sub> measures used would be a useful methodological investigation, although would not currently be possible at the national level.

Third, the PM<sub>10</sub> estimates were derived from a model parameterised to 2001 whereas the mortality data covered 2001 to 2005. This period was chosen to allow for effect latency following exposure, although the more acute effects of PM<sub>10</sub> exposure post-2001 cannot be ruled out. Finally, our models adjusted for the influence of socioeconomic status, ethnicity and smoking at the area level, whilst individual-level measures, if these had been available, may have provided more precise estimates of risk.

## CONCLUSIONS

Our work revealed clear evidence that particulate air pollution is related to adverse health effects in urban New Zealand, and we concluded that modest reductions in PM<sub>10</sub> are likely to have significant health benefits for the population. Increased health risks were even identified below the current level that is considered to be acceptable for human health, suggesting that a lower guideline value should be established and enforced. Contrary to

findings from elsewhere we found little evidence that socioeconomic health inequalities were accentuated in communities with high exposure to PM<sub>10</sub>. Hence deprived communities are not more susceptible to the health effects of air pollution than other groups. Air pollution reductions are therefore only likely to reduce health inequalities if they are greatest in the most deprived areas.

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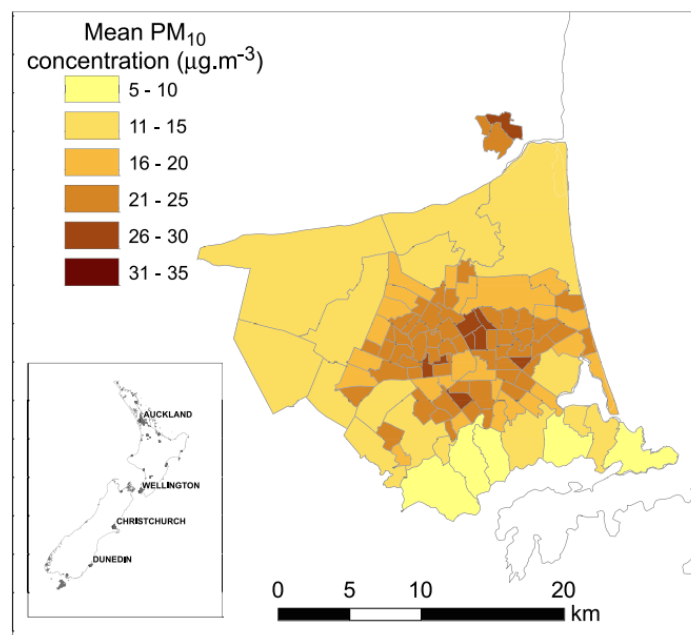
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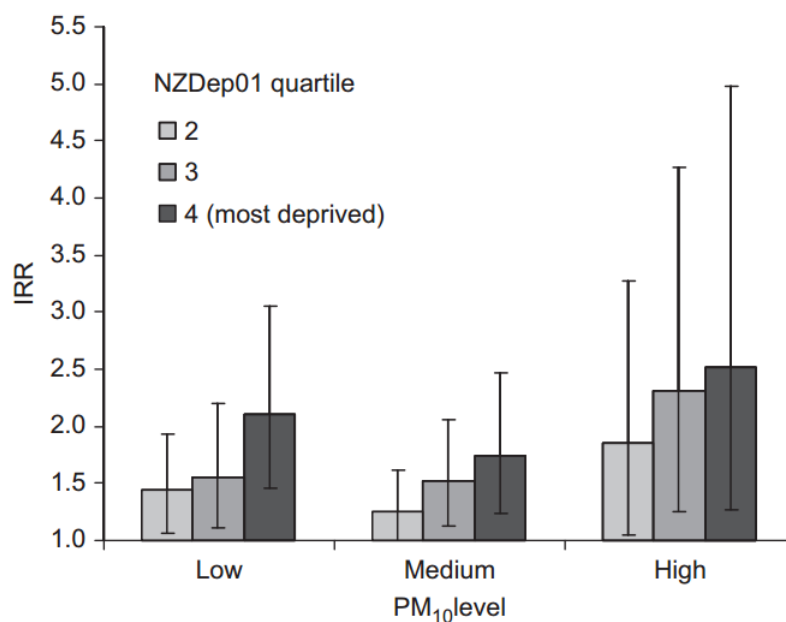
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## FIGURES



**Figure 1. Particulate air pollution in Christchurch, New Zealand.**

Mean annual concentrations of airborne particulate matter with an aerodynamic diameter < 10 µm (PM<sub>10</sub>, µg m<sup>-3</sup>) estimated for Census Area Units.



**Figure 2. Respiratory disease mortality risk by deprivation and air pollution.**

Models were adjusted for individual age-group and sex, and area ethnicity and smoking rate. IRRs are given relative to NZDep01 quartile 1 (least deprived) and error bars indicate 95% confidence intervals. IRR, incidence rate ratio; NZDep01, New Zealand Index of Deprivation 2001; PM<sub>10</sub>, particulate matter with an aerodynamic diameter < 10 µm.

## TABLES

**Table 1.** Incidence rate ratios (+ 95 % confidence intervals) for the association between PM<sub>10</sub> and respiratory disease mortality (plus covariate IRRs).

Variable	Model 1 <sup>a</sup> (Baseline)	Model 2 <sup>b</sup> (+ Area deprivation)	Model 3 <sup>c</sup> (+ Area ethnicity)	Model 4 <sup>d</sup> (+ Area smoking)
<i>PM<sub>10</sub> level</i>				
Low	1.00	1.00	1.00	1.00
Medium	1.17 (1.04 to 1.32)**	1.14 (1.03 to 1.26)*	1.15 (1.04 to 1.27)**	1.15 (1.04 to 1.27)**
High	1.26 (1.09 to 1.47)**	1.19 (1.05 to 1.35)**	1.28 (1.12 to 1.46)***	1.18 (1.03 to 1.35)*
<i>Area deprivation</i>				
1 (least)		1.00	1.00	1.00
2		1.67 (1.43 to 1.97)***	1.55 (1.30 to 1.84)***	1.35 (1.13 to 1.62)**
3		2.41 (2.08 to 2.80)***	2.11 (1.75 to 2.55)***	1.65 (1.34 to 2.03)***
4 (most)		3.46 (2.98 to 4.02)***	2.83 (2.28 to 3.50)***	2.00 (1.58 to 2.54)***
<i>Area % Māori/Pacific</i>				
1 (lowest)			1.00	1.00
2			1.16 (0.99 to 1.35)	1.03 (0.87 to 1.22)
3			1.16 (0.98 to 1.38)	0.98 (0.82 to 1.18)
4 (highest)			1.33 (1.08 to 1.63)**	1.05 (0.85 to 1.30)
<i>Area % smokers</i>				
1 (lowest)				1.00
2				1.32 (1.10 to 1.58)**
3				1.60 (1.30 to 1.97)***
4 (highest)				1.92 (1.52 to 2.41)***

\* 0.05 > p ≥ 0.01; \*\* 0.01 > p ≥ 0.001; \*\*\* p < 0.001

<sup>a</sup> Models included PM<sub>10</sub> level, age group and sex

<sup>b</sup> Models additionally included area deprivation (NZDep2001 quartile)

<sup>c</sup> Models additionally included area ethnicity (% Māori/Pacific population, in quartiles)

<sup>d</sup> Models additionally included area smoking (% regular smokers, in quartiles)



**Table 2.** Incidence rate ratios (+ 95 % confidence intervals) for the association between PM<sub>10</sub> and colorectal cancer mortality (plus covariate IRRs).

	Model 1 <sup>a</sup> (Baseline)	Model 2 <sup>b</sup> (+ Area deprivation)	Model 3 <sup>c</sup> (+ Area ethnicity)	Model 4 <sup>d</sup> (+ Area smoking)
<i>PM<sub>10</sub> level</i>				
Low	1.00	1.00	1.00	1.00
Medium	1.13 (1.02 to 1.25)*	1.09 (0.98 to 1.21)	1.08 (0.98 to 1.20)	1.08 (0.97 to 1.20)
High	1.33 (1.15 to 1.54)***	1.27 (1.10 to 1.48)**	1.18 (1.01 to 1.38)*	1.11 (0.95 to 1.30)
<i>Area deprivation</i>				
1 (least)		1.00	1.00	1.00
2		1.18 (1.02 to 1.35)*	1.24 (1.07 to 1.44)**	1.13 (0.95 to 1.33)
3		1.23 (1.07 to 1.41)**	1.40 (1.17 to 1.67)***	1.18 (0.97 to 1.44)
4 (most)		1.12 (0.96 to 1.29)	1.37 (1.11 to 1.68)**	1.04 (0.82 to 1.32)
<i>Area % Māori/Pacific</i>				
1 (lowest)			1.00	1.00
2			0.98 (0.85 to 1.12)	0.88 (0.76 to 1.02)
3			0.87 (0.73 to 1.02)	0.76 (0.64 to 0.90)**
4 (highest)			0.78 (0.64 to 0.95)*	0.64 (0.52 to 0.80)***
<i>Area % smokers</i>				
1 (lowest)				1.00
2				1.30 (1.10 to 1.55)**
3				1.36 (1.12 to 1.66)**
4 (highest)				1.73 (1.38 to 2.17)***

\* 0.05 > p ≥ 0.01; \*\* 0.01 > p ≥ 0.001; \*\*\* p < 0.001

<sup>a</sup> Models included PM<sub>10</sub> level, age group and sex

<sup>b</sup> Models additionally included area deprivation (NZDep 2001 quartile)

<sup>c</sup> Models additionally included area ethnicity (% Māori/Pacific population, in quartiles)

<sup>d</sup> Models additionally included area smoking (% regular smokers, in quartiles)